In The Drawings:

FIGURE 1 illustrates prolonged monitoring of BoNT/A- and /B-induced inhibition of catecholamine release in chromaffin cells and assessment of their SNAP-25 and Sbr/Cbr contents;

FIGURE 2 illustrates expression of wild-type and mutated SNAP-25 in CHO cells and assessment of cleavage of BoNT/A: subsequent evaluation of their ability to rescue evoked secretion in BoNT/A-pre-treated chromaffin cells;

FIGURE 3 illustrates diagrammatic representation of the point-mutations generated within SNAP-25 and ELISA of their susceptibilities to proteolysis by BoNT/A.;

FIGURE 4 illustrates exocytosis rescued by the introduction of BoNT/A-resistant SNAP-25 into BoNT/A pre-poisoned cells is inhibited by BoNT/E;

FIGURE 5 illustrates BoNT/A proteolytic activity persists in poisoned chromaffin cells for at least 3 weeks: only the protease-resistant SNAP-25 mutant rescues evoked secretion;

FIGURE 6 illustrates truncation and mutation of SNAP-25 to determine the C-terminal residues required for exocytosis;

FIGURE 7 illustrates susceptibility of various SNAP-25 isoforms to BoNT/A, BoNT/C and BoNT/E;

FIGURE 8 illustrates amino acid sequence of human VAMP1, synaptobrevin2, mouse synaptobrevin2, human VAMP3, human SNAP-23, human SNAP-25A, human SNAP-25b and human syntaxin 1A and alignment of human and mouse SNAP-25;

FIGURE 9 illustrates gene therapy as an approach to rescuing neuro-exocythosis in botulinised patients: an example.

DETAILED DESCRIPTION--.